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Research Article

STUDIES OF TOTAL PULMONARY CAPACITY AND ITS SUBDIVISIONS. IV. PRELIMINARY OBSERVATIONS ON CASES OF PULMONARY EMPHYSEMA AND OF PNEUMOCONIOSIS

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There has been no practical method of estimating in a quantitative way the degree of respiratory inefficiency in cases of chronic pulmonary disease. Although many observations have been made in regard to their underlying pathological anatomy and physiology, there has not been any serious attempt to correlate such observations with the clinical condition, especially with respect to adaptation to the increased respiratory demands on physical activity. Investigators have usually been concerned with circulatory and internal respiratory functions. In previous papers (3) (4), we have presented the results of our measurements of capacity of the lungs and of chest expansion in an attempt to establish the normal variation, thus enabling one to distinguish pathological deviations. In the present communication are presented similar observations in cases of chronic pulmonary diseases.

METHODS AND CASES STUDIED

A complete description of the methods used in measuring the total pulmonary air and its subdivisions and in the prediction of their normal values in a given case has been discussed in previous papers (3) (4). Measurements of pulmonary ventilation during physical activity have also been made using a modified bicycle ergometer and a 100-liter Tissot spirometer adapted to the graphic registration of the respirations. In some cases arterial blood was obtained and its oxygen saturation and carbon dioxide content determined by the manometric method of Van Slyke (7). We have studied 15 cases; 9 cases of pulmonary emphysema and 6 cases of pneumoconiosis. Several patients presented evidence of heart disease but no evidence of heart failure was present at the time of these observations. In each group there was considerable variation in the extent of pulmonary disease. This was an important consideration as one of the main purposes of this investigation was to correlate the findings with the degree of functional respiratory disability. A summary of each case is given below:

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Case 1. Male 41 years. Chronic cough for 27 years. Severe asthmatic attacks during the last few years. *Very severe dyspnea on physical activity.* No cardiac symptoms.
Chest markedly emphysematous. Heart normal. Radiograph showed increased linear markings and emphysema.
Diagnosis: Chronic bronchitis. Asthma. Pulmonary emphysema.

Case 2. Male 54 years. Chronic cough and asthmatic attacks for 20 years. *Severe dyspnea on physical activity.* No cardiac symptoms.
Chest markedly emphysematous. Heart normal. Electrocardiogram normal. Radiograph showed increased linear markings.
Diagnosis: Chronic bronchitis. Asthma. Pulmonary emphysema.

Case 3. Male 51 years. Chronic cough, expectoration, and asthmatic attacks for two years. *Severe dyspnea on physical activity.*
No cardiac symptoms.
Chest very emphysematous. Radiograph showed slight left sided cardiac enlargement and increased lung markings. Electrocardiogram presented evidence of myocardial damage.
Diagnosis: Chronic bronchitis. Asthma. Pulmonary emphysema. Chronic myocarditis (compensated).

Case 4. Male 41 years. Chronic cough and asthmatic attacks for 18 years. *Only slight dyspnea on physical activity.* No cardiac symptoms.
Chest not emphysematous in appearance. Hyperresonance on percussion over precordial and hepatic areas. Electrocardiogram normal. Radiograph of the chest showed prominence of the lung marking and of the pulmonary artery suggesting early fibrosis and pulmonic arteriosclerosis.
Diagnosis: Chronic bronchitis. Asthma. Pulmonary emphysema. Pulmonic arteriosclerosis.

Case 5. Male 60 years. Dry cough for many years. *Moderate dyspnea on physical activity.* Frequent attacks of precordial pain for two or three years.
Chest definitely emphysematous in appearance. Heart enlarged to the left. General arteriosclerosis.
Diagnosis: Arteriosclerotic heart disease. Pulmonary emphysema.

Case 6. Male 26 years Asthmatic attacks since childhood. *No dyspnea on physical activity.*
Chest not emphysematous. Heart normal. Radiograph of chest showed increased linear markings and evidence of an early emphysema.
Diagnosis: Asthma. Pulmonary emphysema.

Case 7. Male 41 years. Attacks of precordial pain for seven years. During the last two years attacks of unconsciousness accompanied with convulsions. *No dyspnea on exertion.*
Chest markedly emphysematous. Heart enlarged to the left. Radiograph of the lungs showed pulmonary fibrosis and emphysema.
Diagnosis: Coronary heart disease. Question of Adams-Stokes syndrome. Pulmonary emphysema.

Case 8. Male 50 years. *Slight dyspnea on exertion.*
Chest does not appear emphysematous. Heart enlarged to the left. Electrocardiogram showed left ventricular preponderance and myocardial damage. Radiograph of the chest suggested pulmonary emphysema, pleural thickening and cardiac hypertrophy.
Diagnosis: Arteriosclerotic heart disease. Pulmonary emphysema.

Case 9. Male 61 years. Chronic cough for three years. *Very severe dyspnea* on physical activity. No cardiac symptoms.
Chest markedly emphysematous. Heart enlarged to the left. Electrocardiogram showed an intraventricular conduction defect. Radiograph of the chest revealed a marked degree of emphysema; enlargement of the left ventricle and increased linear markings.
Diagnosis: Chronic bronchitis. Arteriosclerotic heart disease. Pulmonary emphysema.

Case 10. Male 48 years. Had worked in sand blasting for 6 years. Chronic cough for four years. *Severe dyspnea on physical activity.* No cardiac symptoms.
Chest emphysematous. Heart not enlarged. Electrocardiogram gave questionable evidence of myocardial damage. Radiograph of the chest showed marked increase in linear markings and typical appearance of pneumoconiosis and emphysema at both bases.
Diagnosis: Pneumoconiosis. Pulmonary fibrosis.

Case 11. Male 35 years. Had worked at sand-blasting for 5 years. Chronic cough for two years. *Moderate dyspnea on exertion.* Able to carry on light work. Complains of palpitation on physical activity.
Chest did not appear emphysematous. Heart not enlarged. Radiograph of the chest showed characteristic changes of pneumoconiosis, pulmonary fibrosis and emphysema. Electrocardiogram normal.
Diagnosis: Pneumoconiosis. Pulmonary fibrosis.

Case 12. Male 46 years. Had worked at sand-blasting for 2 years. Slight cough during the last year. *Moderate dyspnea on physical activity.* No cardiac symptoms.
Chest had normal appearance. Heart not enlarged. Electrocardiogram normal. Radiograph of the chest suggested pneumoconiosis and pulmonary emphysema.
Diagnosis: Pneumoconiosis. Pulmonary fibrosis.

Case 13. Male 42 years. Had been exposed to inhalation of siliceous dust for 18 months. *Moderate dyspnea on exertion.* No other symptoms.
Chest had emphysematous appearance. Heart slightly enlarged to the left. Electrocardiogram indicated left ventricular preponderance. Radiograph of the chest showed changes consistent with pneumoconiosis (Early second stage).
Diagnosis: Pneumoconiosis. Pulmonary fibrosis. Myocardial disease (compensated).

Case 14. Male 42 years. Had worked at sand-blasting for 8 months. Chronic cough during the last two years. He thought that in the last two months he had had *slight dyspnea on physical activity.* No other symptoms.
Chest normal. Heart not enlarged. Radiograph of the chest showed increase in size of the hila and increased markings but no mottling. Appearance of emphysema in both bases.
Diagnosis: Question of pneumoconiosis. Question of pulmonary fibrosis.

Case 15. Male 56 years. Had been severely exposed to inhalation of siliceous dust 7 years. Hemoptysis, frequent cough and *severe dyspnea on exertion.* Edema of lower extremities.
Chest markedly emphysematous. Heart greatly enlarged. Electrocardiogram showed right ventricular preponderance. Radiograph of

the lungs revealed a diffuse soft mottling. Pulmonary artery prominent. The total volume of the blood was increased.
Diagnosis: Pneumoconiosis. Pulmonary fibrosis. Sclerosis of the pulmonary artery. Myocardial disease. Secondary polycythemia.

Observations on pulmonary capacity

Pulmonary emphysema. The calculated and the observed values for the total pulmonary air and its main subdivisions in the nine subjects having pulmonary emphysema are presented in Table 1, and are shown in Figure 1. In these cases the total capacity of the lungs which was ob-

TABLE 1

Comparison of observed pulmonary capacity with calculated normal values in emphysema and in pneumoconiosis

Case number	Total capacity			Vital capacity			Mid capacity			Residual air		
	Calculated	Observed	Difference	Calculated	Observed	Difference	Calculated	Observed	Difference	Calculated	Observed	Difference
	liters	*liters*	*per cent*	*liters*	*liters*	*per cent*	*liters*	*liters*	*per cent*	*liters*	*liters*	*per cent*
						Pulmonary emphysema						
1	8.53	6.64	−22.2	6.66	3.05	−54.2	3.24	4.67	+ 44.1	1.89	3.59	+ 90.0
2	7.25	8.52	+17.5	5.66	2.70	−52.3	2.75	6.56	+138.5	1.59	5.82	+266.0
3	5.62	5.96	+ 6.0	4.39	1.42	−67.7	2.13	5.32	+149.0	1.23	4.54	+269.1
4	6.89	7.59	+10.1	5.38	4.95	− 8.0	2.62	3.72	+ 41.9	1.51	2.64	+ 74.8
5	6.47	6.18	− 4.5	5.05	4.00	−20.8	2.46	3.56	+ 44.7	1.42	2.18	+ 53.5
6	5.02	5.76	+14.7	3.92	3.64	− 7.2	1.91	2.96	+ 54.9	1.10	2.12	+ 92.7
7	6.20	5.08	−18.1	4.84	3.60	−25.7	2.36	1.96	− 17.0	1.36	1.48	+ 8.8
8	5.70	4.32	−24.3	4.45	2.98	−33.1	2.17	1.66	− 23.5	1.25	1.34	+ 7.2
9	5.98	5.04	−15.8	4.67	1.44	−69.2	2.27	4.08	+ 79.7	1.31	3.60	+174.8
						Pneumoconiosis						
10	6.56	3.30	−49.7	5.12	1.44	−71.9	2.49	2.22	− 10.8	1.44	1.86	+29.1
11	5.11	3.63	−29.0	3.99	2.32	−41.9	1.94	1.85	− 4.7	1.12	1.31	+16.9
12	6.05	4.65	−23.2	4.72	2.86	−39.5	2.30	2.53	+ 10.0	1.33	1.79	+34.5
13	4.96	4.24	−14.8	3.87	2.84	−26.7	1.88	1.84	− 2.2	1.09	1.40	+28.4
14	5.53	5.06	− 8.5	4.32	3.90	− 9.8	2.10	1.70	− 19.1	1.21	1.16	− 7.2
15	6.82	4.04	−40.8	5.32	2.08	−60.6	2.59	2.28	− 12.0	1.50	1.96	+30.6

served corresponds with that calculated from measurements of the radiograph of the chest, as was the case with normal men. When we analyze the subdivisions of the pulmonary air we find, however, very significant differences. The vital capacity is markedly reduced in emphysema and this is associated with a great increase in the volume of residual air. The degree of this change was closely correlated with the severity of the patients' symptoms. The volume at mid capacity is, in most cases, much greater than the normal value predicted from radiographic measurements.

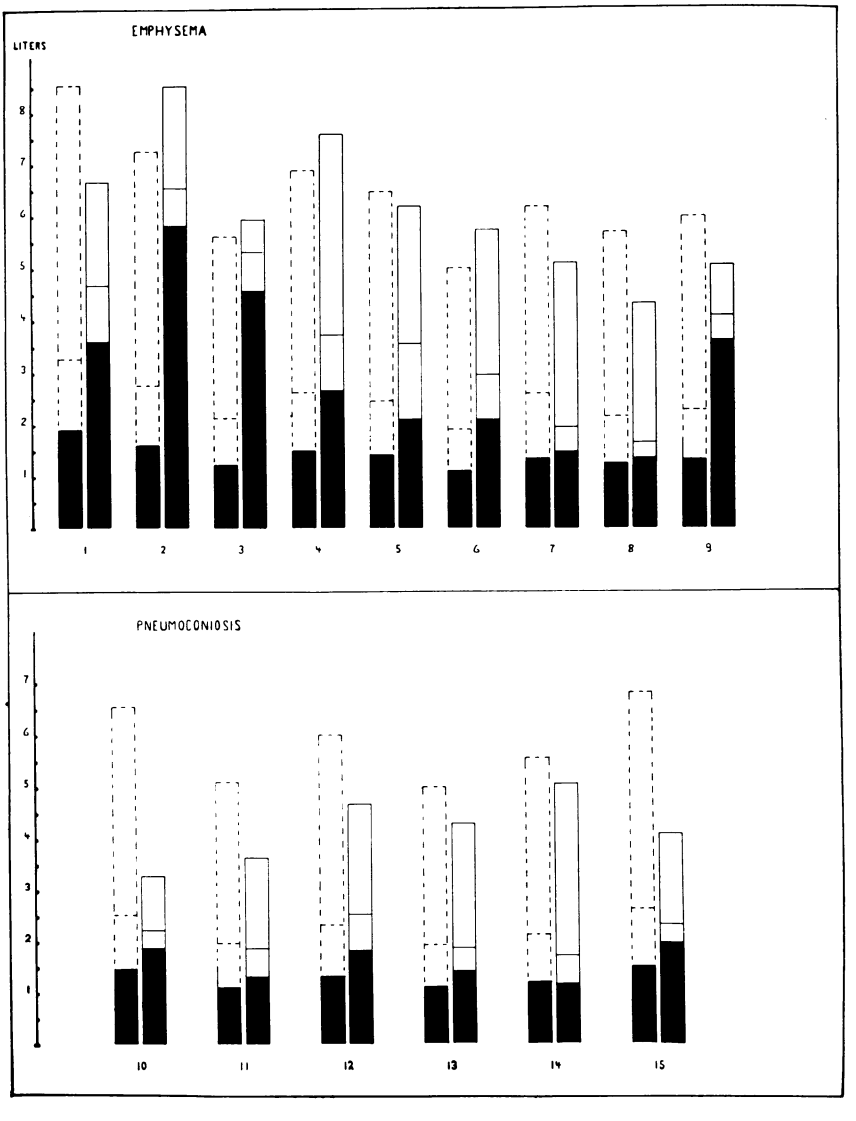

FIG. 1. CALCULATED AND OBSERVED PULMONARY CAPACITY IN CASES OF PULMONARY EMPHYSEMA AND PNEUMOCONIOSIS

Each case is represented by two columns. The one on the left, with broken lines, is the predicted value, and the right column is the observed volume.

In black, residual air. Transverse line dividing the white area (vital capacity) is the mid capacity level.

These alterations in the absolute values of the pulmonary airs are reflected in the relative values. That this is so may be appreciated by a study of Table 2. The vital capacity is reduced and the residual air correspondingly increased. In severe cases the residual air occupies a much greater proportion of the total volume than the vital capacity. A profound alteration in the ability to ventilate the alveoli has accordingly taken place.

TABLE 2

Relative values of subdivisions of pulmonary capacity in emphysema and pneumoconiosis

Case number	Ratio $\frac{\text{Vital capacity}}{\text{Total volume}} \times 100$		Ratio $\frac{\text{Residual air}}{\text{Total volume}} \times 100$		Ratio $\frac{\text{Mid capacity}}{\text{Total volume}} \times 100$		Ratio $\frac{\text{Complementary air}}{\text{Vital capacity}} \times 100$	
	Normal	Observed	Normal	Observed	Normal	Observed	Normal	Observed
Pulmonary emphysema								
1	78.0	45.9	22.0	54.1	38.0	70.4	79.4	64.6
2	78.0	31.7	22.0	68.3	38.0	77.0	79.4	72.6
3	78.0	23.8	22.0	76.4	38.0	87.6	79.4	45.1
4	78.0	65.2	22.0	34.8	38.0	49.1	79.4	78.2
5	78.0	64.7	22.0	35.3	38.0	57.6	79.4	65.5
6	78.0	63.2	22.0	36.8	38.0	51.4	79.4	77.0
7	78.0	70.8	22.0	29.1	38.0	38.5	79.4	88.1
8	78.0	64.3	22.0	35.7	38.0	38.7	79.4	89.3
9	78.0	28.5	22.0	71.4	38.0	80.9	79.4	66.6
Pneumoconiosis								
10	78.0	43.6	22.0	56.4	38.0	67.3	79.4	75.0
11	78.0	63.7	22.0	36.0	38.0	50.9	79.4	76.7
12	78.0	61.5	22.0	38.4	38.0	54.4	79.4	79.7
13	78.0	69.3	22.0	30.7	38.0	43.4	79.4	84.5
14	78.0	77.1	22.0	22.9	38.0	33.6	79.4	86.1
15	78.0	51.4	22.0	48.6	38.0	56.5	79.4	84.6

In many cases, especially in the severe ones, the complementary air makes up a smaller portion of the vital capacity than is the case with normal persons. This defect is a noteworthy feature in Cases 1 and 3, in which the volume of reserve air is strictly normal. It appears therefore that the distended lungs cannot be deflated to the normal level, but that a forced expiration expels nevertheless a normal quantity of air, owing to the change in volume of mid capacity.

When the vital capacity is composed chiefly of reserve air, or when the latter volume is not as markedly reduced as the complementary air, there is probably a further handicap to effective alveolar ventilation.

Christie (2) found the volume of the reserve air was smaller after a maximum inspiration than is the case when it is measured after a normal expiration and interpreted this as an indication of diminished elasticity of

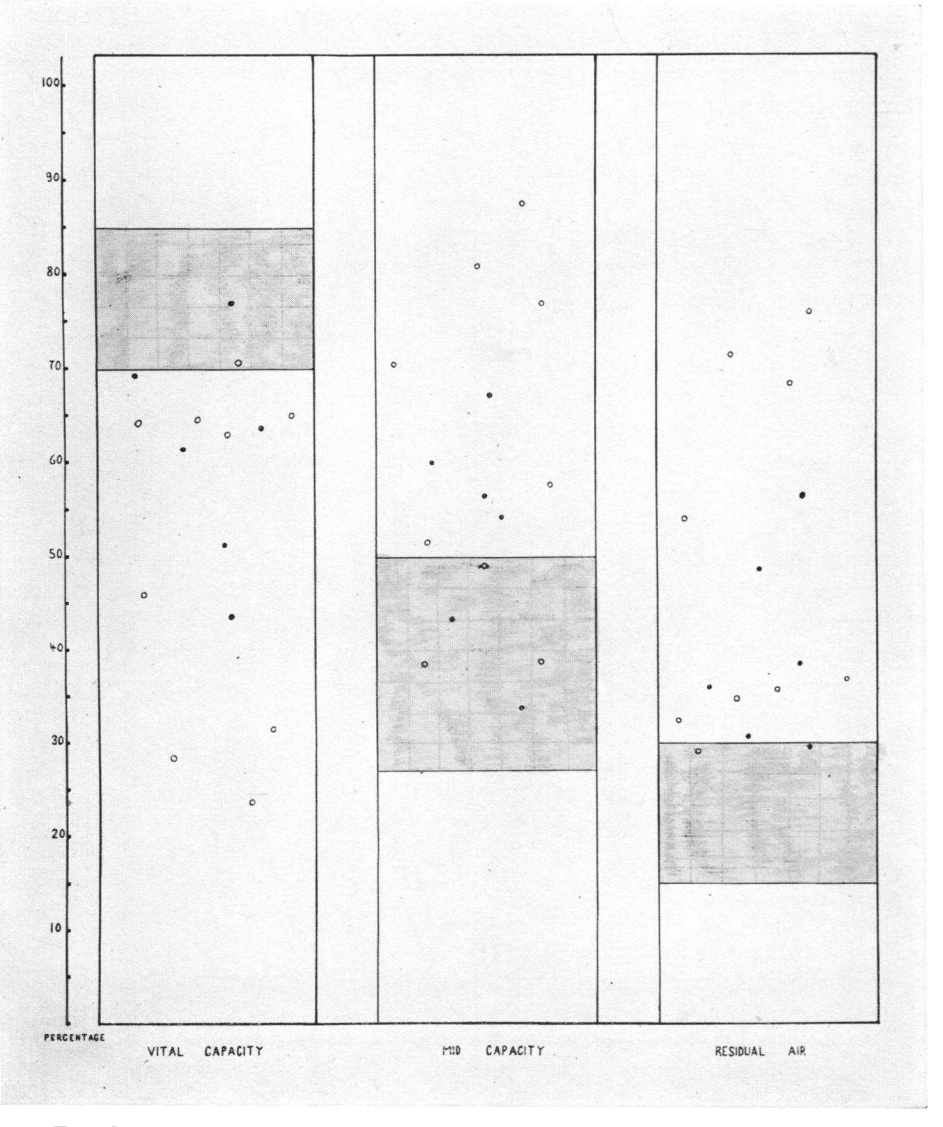

FIG. 2. RELATIVE PULMONARY CAPACITIES (TOTAL CAPACITY = 100 PER CENT) IN CASES OF PULMONARY EMPHYSEMA AND PNEUMOCONIOSIS

The shaded areas represent limits of normal variations. Black dots are cases of pulmonary emphysema; circles are cases of pneumoconiosis.

the lungs in pulmonary emphysema. There is, apparently, a decrease in the ability of individuals to deflate the lungs after they have been fully expanded. We have investigated this phenomenon in all our cases and have found that it is not always present. It occurred in two instances but was not evident in some of the most severe and most unquestionable cases of emphysema.

Pneumoconiosis. The results of our observations in this condition differ from those obtained in pulmonary emphysema. The observed total capacity of the lungs is as a rule lower than the calculated. This decrease is proportional to the clinical and radiological evidence of the severity of the disease. The lungs of these patients contain, at maximum inspiration, a smaller volume of air than would be predicted from the radiographic measurements of the chest. The diminution in total capacity is caused entirely by decrease in the vital capacity. The residual air is moderately increased in almost all cases, and this change indicates that there is some degree of pulmonary emphysema associated with the fibrotic changes. This observation agrees with the frequent demonstration by pathologists and radiologists of emphysematous areas in the lungs of these patients. The mid capacity is found to be normal in most instances. The vital capacity is reduced in proportion to the total volume, and the residual air greater, but these alterations are relatively less than in cases of pulmonary emphysema. The increase in the ratio (Residual air/Total volume) is chiefly due to diminution in the vital capacity. The two components of the vital capacity, the reserve and complementary air, show a normal relationship to each other in pneumoconiosis.

Chest expansion

Changes in the ratio (Area at maximum expiration/Area at maximum inspiration) $\times$ 100 (measured on the doubly exposed radiograph of the lungs) are especially interesting. From previous studies (4) of normal male subjects we concluded that a ratio higher than 72.0 may be considered as indicating diminished expansion. In all but two of the cases of pulmonary emphysema this ratio was higher than 72 (Table 3). In these two the diagnosis of emphysema was made on clinical grounds only. In neither of them did the pulmonary capacity show marked significant abnormality. It appears then that in undoubted cases of pulmonary emphysema there is a conspicuous decrease in ability to expand the chest (see Figure 3). The diaphragmatic excursion and the lateral expansion of the chest also are as a rule decreased. The angle of movement of a rib in changing from full expiration to maximum inspiration was markedly reduced in the majority of patients. In two of the severest cases movement was almost nil.

In pneumoconiosis the ratio (Area at maximum expiration/Area at maximum inspiration) $\times$ 100 was within upper normal limits in all but two cases. In one it was markedly altered, due to reduced expansion of

TABLE 3

Chest expansion

Case number	Ratio $\frac{\text{Area at maximum expiration}}{\text{Area at maximum inspiration}} \times 100$	Diaphragmatic excursion *	Lateral expansion	Rib movement	Circumference expansion
		cm.	*cm.*	*degrees*	*cm.*
		Pulmonary emphysema			
1	90.9	0.3	2.3	20	4.0
2	86.8	3.3	2.2	7	4.5
3	84.9	2.4	0.4	3	2.0
4	77.9	5.8	2.8	14	10.0
5	70.7	5.1	1.0	11	8.0
6	75.5	4.3	2.2	22	7.0
7	77.4	3.3	1.0	12	8.0
8	66.9	4.7	0.6	10	5.0
9	82.9	4.8	0.2	2	3.0
		Pneumoconiosis			
10	89.5	0.8	1.8	8	4.0
11	70.0	5.0	2.5	15	6.0
12	73.5	4.7	0.9	13	5.0
13	60.6	5.3	2.8	18	4.0
14	70.9	4.1	2.3		6.0
15	73.8	5.3	1.1	3	3.5

* Average of both diaphragms.

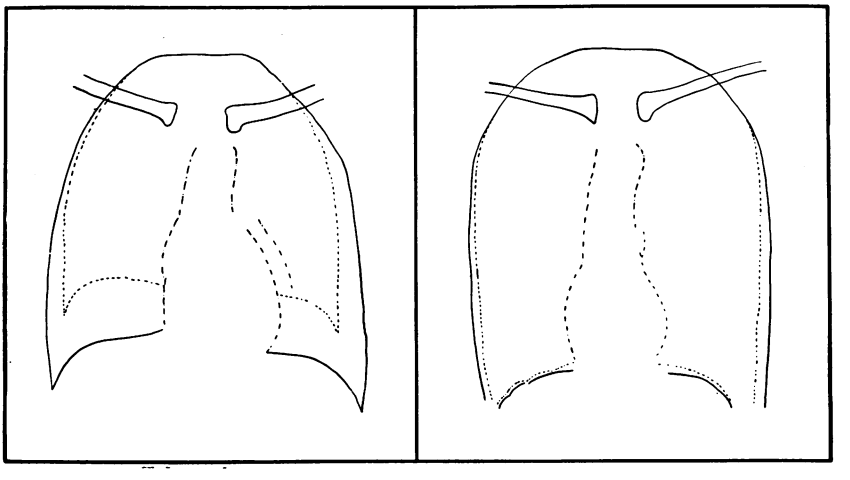

FIG. 3. OUTLINE OF THE LUNG FIELDS OF MAXIMUM INSPIRATION AND EXPIRATION IN A NORMAL MAN (LEFT) AND IN A CASE OF PULMONARY EMPHYSEMA (RIGHT).

the chest. The diaphragmatic excursion, the lateral expansion of the chest and the movement of the rib were usually within normal limits. Reduction in chest expansion is not characteristic of pneumoconiosis and pulmonary fibrosis, as it is of pulmonary emphysema.

Relationship of dyspnea to alterations in capacity of the lungs

A striking correlation is found to exist between the observed alterations in capacity of the lungs and the tendency to dyspnea. In all those cases in which there was severe limitation in physical activity we find marked changes in the absolute and relative pulmonary capacity. A high ratio (Residual air/Total volume) is almost always accompanied by a pronounced degree of dyspnea on physical exertion. On the other hand, in Cases 4, 5, 7, 8 and 14, in which such a ratio was either normal or but slightly increased the history of dyspnea on exertion was not conspicuous. The tendency to dyspnea in the cases of pneumoconiosis was less severe, as a rule, than in those with pulmonary emphysema, and the changes in the relative pulmonary capacities were correspondingly not as great. In two cases in which the ratio of residual air to total volume was high dyspnea on physical activity was, however, also severe.

While discussing the adaptation to physical activity it will be pertinent to present some observations on the measurement of pulmonary ventilation during exercise. The observations included graphic registration of the number of respirations, tidal and minute volumes, of patient while engaged in work in a specially designed chair ergometer. These investigations are summarized graphically in Figure 4. Normally the process of adaptation to physical activity necessitates greater frequency of respiration as well as increase in the tidal volume, so that the ventilation per minute is considerably increased. Peabody (6) found that efficient ventilation during exercise is closely related to the vital capacity, and that when this is low the maximum possible pulmonary ventilation is reduced proportionally. In pneumoconiosis, in which there is a decrease in the vital capacity, patients were able to increase the number as well as the depth of the respirations during exercise yet the total ventilation was considerably less than in men. In pulmonary emphysema an abnormal response to physical exercise is evident. The vital capacity was low and there was diminished ability to expand the chest. Emphysematous individuals during physical activity tend to increase the rate rather than the depth of breathing (Fig. 4). Difficulty in expansion of the chest in emphysema is so great that when accessory muscles are brought into play voluntarily increase in the depth of breathing is accompanied by decrease in rate. In all the cases subjected to exercise tests there has been severe dyspnea on exertion, and in each case the maximum ventilation attainable has been less than normal.

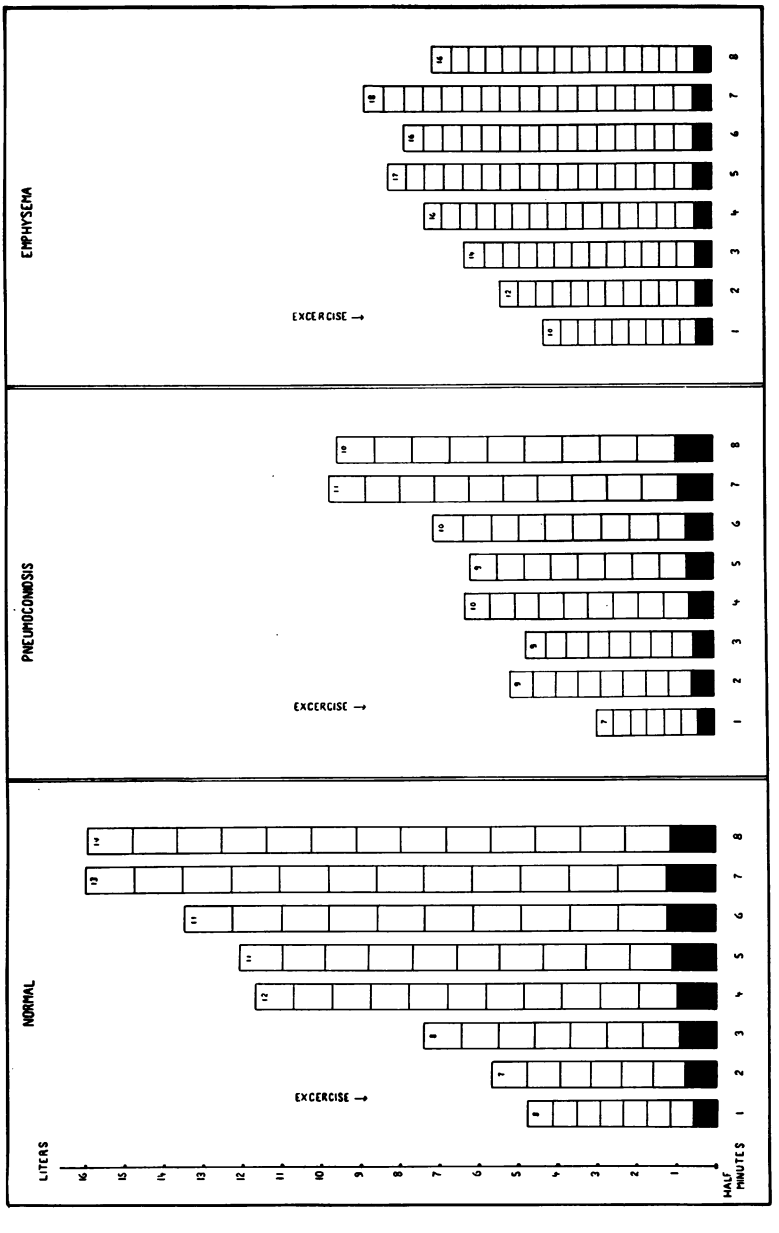

FIG. 4. VENTILATION DURING EXERCISE. DIAGRAM CONSTRUCTED FROM SEVERAL OBSERVATIONS MADE ON NORMAL MALE INDIVIDUALS, AND OF CASES OF PNEUMOCONIOSIS AND PULMONARY EMPHYSEMA.

Height of each column represents the total ventilation during succeeding half minutes. Each subdivision in the column represents the tidal volume and the figure within the column the number of respirations during that half minute.

Pulmonary capacity and arterial saturation with oxygen

In seven cases the arterial blood has been examined to determine its content of carbon dioxide and the degree of saturation with oxygen. The results and the corresponding pulmonary capacities are presented in Table 4. The lowest values for saturation of arterial blood with oxygen have been found in cases in which the ratio (Residual air/Total volume) was abnormally high.

TABLE 4

Relationship of arterial oxygen saturation to pulmonary capacity

Cases	Oxygen saturation arterial blood	CO_2 content arterial blood volume	Decrease in vital capacity	Increase in residual air	Ratio $\frac{\text{Residual air}}{\text{Total volume}}$
	per cent	*per cent*	*per cent*	*per cent*	*per cent*
(3)	72.9	49.3	−67.7	+269.1	76.2
(1)	79.6	47.6	−54.2	+90.0	54.1
(2)	80.0	46.7	−52.3	+266.0	68.3
(10)	80.6	55.2	−71.9	+29.1	56.4
(11)	81.2	46.2	−41.9	+16.9	36.3
(4)	95.6	50.2	− 8.0	+74.8	34.8
(12)	95.7	44.2	−39.5	+34.5	38.5

DISCUSSION

Marked and significant changes in the absolute and relative pulmonary capacities exist as these studies show, in cases of pulmonary emphysema. The results are in agreement with the investigations of Lundsgaard and Schierbeck (5) and Anthony (1), who found a normal total volume but a marked decrease in the vital capacity and a corresponding increase in the residual air. The fact that there are definite alterations in the capacity of the lungs in cases of this nature, and that these changes are closely correlated with the clinical condition and the tendency to dyspnea, is of considerable importance. The diagnosis of pulmonary emphysema is not so easy to establish clinically as it appears to be. An emphysematous appearance of the chest may be due entirely to skeletal changes without alteration in the lungs, and conversely actual emphysema, in the pathological-physiological sense of the word, may be present and may escape clinical detection because the chest is of normal size and shape. Not infrequently symptoms of respiratory failure are attributed to heart disease, especially in people past middle age, when pulmonary changes alone are responsible for the symptoms. In doubtful cases the measurement of the pulmonary capacity will aid in establishing the correct diagnosis. The calculated pulmonary capacity in some cases of pulmonary emphysema is not strictly to be regarded as 'normal,' since we are using as a basis for prediction the size of the chest which in itself may be pathological. To be normal the capacity of the lungs in a given case must correspond to the volume of

the chest before pathological alterations occurred. We have investigated in normal men the possible correlation of thoracic volume with other bodily characteristics, and have not found any correlation by means of which we could predict normal values from other data, such as height, weight, or surface area. In emphysema observed total pulmonary capacities agree fairly closely with the size of the chest estimated from measurements of radiographs. The most significant alterations occur in the relative values of the subdivisions of total capacity.

There are no previous observations in the literature regarding pulmonary capacity in cases of pneumoconiosis and pulmonary fibrosis. We have observed that there are significant changes in such cases, the degree of which is closely correlated with the functional efficiency of respiration and with the anatomical changes revealed in the radiological examinations. From these observations it seems probable that the determination of the pulmonary capacity may be of value in the proper evaluation of the degree of respiratory disability. In Case 14 in which the evidence of fibrosis in the lungs was small, the pulmonary capacity was normal. But in Cases 10 and 11, in which it was great the capacity of the lungs was much diminished. In four of five cases there was moderate increase in the volume of residual air, due probably to secondary emphysema usually found in association with advanced pulmonary fibrosis.

SUMMARY AND CONCLUSIONS

1. In seven of nine cases of pulmonary emphysema the total pulmonary capacity observed corresponded closely with that predicted from measurements of the chest cavity. In two it was slightly less. Increase in the volume of the residual air, and a corresponding reduction in the vital capacity was observed in all cases.

2. In emphysematous patients there was definite reduction in expansion of the chest, the degree being closely correlated with alterations in the relative pulmonary capacities.

3. In cases of pneumoconiosis (pulmonary fibrosis) the total capacity of the lungs observed was less than that predicted from measurements of the chest, due to decrease in the vital capacity. The residual air was moderately increased in four of five cases. In one the changes were minimal.

4. Decrease in expansion of the chest was not a significant feature of cases of pneumoconiosis (pulmonary fibrosis).

5. Cases in which the ratio (Residual air/Total volume) was abnormally high were found to exhibit low saturation of the arterial blood with oxygen, indicating poor alveolar ventilation.

Preliminary observations on response to exercise showed that the capacity to ventilate the lungs was limited in a severe case of emphysema, and in one of pneumoconiosis, compared with that in a normal man.

Further observations will be necessary to establish a relation between the degree of functional disability and abnormalities in pulmonary capacity.

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